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Original article

Application of neutrophil/lymphocyte ratio in predicting coronary blood flow and mortality in patients with ST-elevation myocardial infarction undergoing percutaneous coronary intervention

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ABSTRACT

Background: To investigate the potential correlation of neutrophil/lymphocyte ratio (NLR) to coronary blood flow and in-hospital along with long-term mortality in patients with ST-elevation myocardial infarction (STEMI) undergoing percutaneous coronary intervention (PCI).

Methods: In the current study, 636 consecutive patients with STEMI were recruited and stratified into three tertiles by NLRs upon admission (tertile I < 3.0, tertile II 3.0–6.40, tertile III > 6.40). The coronary blood flow was expressed by corrected TIMI frame count (CTFC). The in-hospital mortality and 12-month long follow-up data were collected. Receiver operating characteristic (ROC) curves were also constructed.

Results: Our analysis demonstrated that NLR was positively correlated to CTFC and in-hospital mortality ($r = 0.517$, $p < 0.001$; $r = 0.439$, $p < 0.001$). In the multiple logistic regression analysis, NLR was testified as an independent risk factor for coronary blood flow after PCI and in-hospital mortality [odds ratio (OR) = 2.031, 95% confidence interval (CI): 1.627–2.435, $p < 0.001$; OR = 1.176, 95% CI: 1.025–1.351, $p = 0.021$]. During the 12-month follow-up, there were a total of 43 deaths and statistically significant increase in long-term mortality was observed in patients from tertile I to III ($p = 0.005$). In the ROC curves analysis, the area under the curve (AUC = 0.607, 95% CI: 0.475–0.739, $p = 0.253$), with threshold value of 5.9 (sensitivity: 63.7%, specificity: 61.1%) for predicting in-hospital mortality.

Conclusions: NLR, an indicator that can be tested in the laboratory with low cost and time consumption, is independently correlated to coronary blood flow and acts as an independent risk factor for in-hospital mortality in patients with STEMI undergoing PCI.

Introduction

As one of the diseases with high morbidity and mortality in the world, cardiovascular disease (CVD), especially acute myocardial infarction (AMI) has a serious impact on life expectancy and life quality of human beings [1]. AMI is usually due to ischemia of the myocardium caused by coronary artery occlusion. The main diagnosis of AMI is by electrocardiogram (ECG) while ST-elevation myocardial infarction (STEMI) is necrosis of the myocardial tissue caused by total occlusion of a coronary artery, which can be indicated by persistent elevation of the ST-segment on ECG. As for prognosis, scoring systems such as the thrombolysis in myocardial infarction (TIMI) score [2], the Platelet glycoprotein IIb/IIIa in Unstable agina: Receptor Suppression Using Integrilin (PURSUIT) score [3], and the Global Registry of Acute Coronary Events (GRACE) score [4] have been adopted to allow clinicians to stratify risk of disease. There is still a lack of prognostic biomarkers to complement the prognostic value of the simple risk scores and the only accepted one is cardiac troponin [5]. It has been well documented that atherosclerosis is the pathological basis of AMI and inflammation plays an important role in the indication and progression of atherosclerosis [6,7]. Given the association of inflammation with AMI, we should devote much effort to discover AMI-associated inflammatory markers. Neutrophil to lymphocyte ratio (NLR) is a widely used inflammatory marker and its linkage to

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coronary heart disease has been recently described [8,9]. An elevated NLR was reported to have implications in long-term mortality as well as impaired coronary arteries in patients with AMI [10–15]. Here in the current study, by recruiting a large sample size with 12 months of clinical follow-up, we have systematically determined the association of NLR with coronary blood flow as well as in-hospital and long-term mortality in patients with STEMI undergoing percutaneous coronary intervention (PCI).

Materials and methods

Ethical statement

Informed written consent was obtained from all patients involved in the study. All protocols involving human subjects were approved by the local Ethics Committee.

Study population

A total number of 636 consecutive patients with the diagnosis of STEMI (78% male, mean age 59.27 ± 11.27 years) admitted to the First Affiliated Hospital of Harbin Medical University from January 2008 to September 2011 were recruited. STEMI was defined as typical chest pain ≥ 30 min with ST-segment elevation > 1 mm in ≥2 consecutive leads on ECG or new-onset left bundle brunch block. Exclusion criteria included treatment with thrombolytic drugs in previous 24 h, active infections, previously proved systemic inflammatory disease, patients with shock or old myocardial infarction, or with history of PCI/coronary artery bypass grafting, cancer, end-stage liver or renal failure, rheumatoid immune system disease, and recent use of steroid drugs. Detailed information on all patients was also collected, including medical history as well as results from physical examination and the laboratory examination (i.e. routine blood test, routine urine test, biochemical series, ECG, transthoracic echocardiography).

NLR calculation

The blood sample was taken immediately after admission and sent for laboratory analysis. The white blood cell (WBC) count as well as subtypes was measured by automatic blood cell analyzer (Ruby, Abbott Laboratories, Abbott Park, IL, USA). NLR was obtained via dividing neutrophil count by lymphocyte count.

PCI procedure and echocardiography

The standard femoral approach with a 7-French guiding catheter was adopted to perform all primary PCI, as previously described [16]. The patients were first injected intravenously with 5000 IU of heparin (70 U/kg) and a 300 mg loading dose of both aspirin and clopidogrel, and then direct stenting or balloon predilatation was performed. The operator determined the choice of stents (bare metal or drug-eluting stent). Transthoracic echocardiography was performed with standard echocardiography procedure using the iE33 xMATRIX Echocardiography System (Philips, Andover, MA, USA) for each patient immediately following PCI in the intensive cardiac care unit. The PCI procedure was successfully performed with all the patients, and stents were successfully implanted with all the patients. The information on patients about the duration (hour) from symptom onset to recanalization with each group was recorded.

Measurement of coronary blood flow

In the present study, coronary blood flow was calculated by the corrected TIMI frame count (CTFC) which was assessed by three independent interventional doctors, and the CTFC was measured at 90–120 min after thrombolytic administration. In addition, nicorandil or nitroprusside was injected into coronary artery if the coronary flow was slow after ballooning. The first and last frames used for TIMI frame counting were defined as follows: in the first frame, contrast medium fully entered the artery and there must be antegrade motion, while in the last frame, contrast medium went into the marked target artery distal branch. The branch of the left anterior descending coronary artery (LAD) used for TIMI frame counting was determined as follows: the distal-most branch in the LAD (also referred to as the "pitchfork," "mustache," or "whale's tail") usually occurred at the apex of the heart. In a wraparound LAD, the branch closest to the apex of the heart was used. The branch of the left circumflex (LCX) used for TIMI frame counting was determined as follows: the artery used for TIMI frame counting was the artery with the longest total distance along which contrast medium traveled in the LCX system but yet passed through the culprit lesion. The branch of the right coronary artery (RCA) used for TIMI frame counting was determined as follows: the distal landmark was the first branch arising from the posterior lateral extension of the RCA after the origin of the posterior descending artery, regardless of the size of this branch. Count of the anterior descending and circumflex selected the right anterior oblique plus foot. Count of the right coronary artery selected left anterior oblique position plus head. The left LAD was longer than the mean of the RCA and LCX. Therefore, the longer LAD frame counts were corrected via dividing by 1.7 to derive CTFC [17].

Follow-up data collection

In-hospital mortality was obtained for all subjects based on hospital records. Survival data post hospital discharge were obtained by a 12-month in person or telephone interview with patients, families, or primary physicians.

Statistical analysis

Patients were categorized into tertiles on the basis of NLR as previously described [11,14]. Continuous variables were tested for normal distribution by Kolmogorov–Smirnov test. Continuous data were presented as mean ± SD and Student's *t* test and Mann–Whitney *U* test were adopted for statistical comparisons between two groups for normally and abnormally distributed data, respectively. Categorical variables were summarized as percentages and chi-square test was applied for statistical comparisons. A multivariate logistic regression model was used to assess the independent association of NLR with CTFC and in-hospital mortality. Odds ratio (OR) with 95% confidence intervals (95% CI) were calculated. A Kaplan–Meier curve was used to plot the 12-month outcomes of the tertiles, and a log-rank test was applied to assess significance across the tertiles. Receiver operating characteristic (ROC) curves were performed to reveal threshold value of NLR that could be used to predict mortality. A p-value < 0.05 was considered statistically significant. Statistical comparisons were performed using SPSS version 18 (SPSS Inc., Chicago, IL, USA).

Results

Basic characteristics of the patients

Of all the patients recruited in the study, 636 cases (100%) and 546 cases (86%) were available for in-hospital and 12-month follow-up data, respectively. Patients' baseline characteristics are listed in Table 1. The patients were stratified into three tertiles according to their NLRs upon admission (tertile I < 3.0, tertile II 3.0–6.40, tertile III > 6.40). There were 212 patients in each tertile with mean age 59.50 ± 11.17 years, 78.8% men in tertile I, mean age

Table 1
Basic characteristics of patients.

Variable	Tertile			
	I (<3.0) (n = 212)	II (3.0–6.40) (n = 212)	III (>6.40) (n = 212)	p value
Age (years)	59.50 ± 11.17	59.22 ± 11.29	59.13 ± 12.00	0.941
Men	167 (78.8%)	163 (76.9%)	165 (77.8%)	0.896
Duration from symptom onset to recanalization (h)	4.830 (3.170–9.628)	5.500 (4.000–9.500)	6.460 (4.500–10.628)	0.614
Hypertension	95 (44.8%)	111 (52.4%)	103 (48.6%)	0.299
Diabetes mellitus	31 (14.6%)	31 (14.6%)	38 (17.9%)	0.320
Smoking	121 (58.7%)	138 (65.1%)	120 (58.3%)	0.277
Drinking	32 (15.1%)	28 (13.2%)	27 (12.7%)	0.756
Systolic pressure on admission (mmHg)	131.72 ± 26.30	129.09 ± 25.53	125.51 ± 25.11	0.048
Heart rate on admission (min^{-1})	71.02 ± 16.44	73.94 ± 19.30	80.11 ± 20.56	<0.001
Peak CK-MB (U/L)	164.17 ± 137.02	204.25 ± 178.83	261.17 ± 226.61	<0.001
Hs-CRP (mg/L)	25.58 ± 26.62	32.39 ± 39.36	44.12 ± 53.16	<0.001
GFR (mL/min/1.73 m^2)	87.53 ± 22.29	86.79 ± 22.24	88.03 ± 25.57	0.864
LVEF on admission (%)	51.50 ± 8.98	50.35 ± 9.60	49.31 ± 9.21	0.055
Total cholesterol (mmol/L)	5.04 ± 1.17	5.01 ± 1.21	4.76 ± 1.08	0.026
Total triglyceride (mmol/L)	1.87 ± 1.20	1.67 ± 0.87	1.65 ± 1.07	0.053
HDL (mmol/L)	1.15 ± 0.26	1.19 ± 0.27	1.19 ± 0.31	0.172
LDL (mmol/L)	3.37 ± 1.15	3.33 ± 1.21	3.02 ± 0.96	0.003
Lipoprotein a (mg/dL)	28.15 ± 19.13	26.40 ± 19.16	29.81 ± 21.11	0.229
WBC ($\times 10^9$/L)	9.21 ± 2.61	10.42 ± 2.96	12.00 ± 3.48	<0.001
Neutrophil ($\times 10^9$/L)	5.35 ± 1.69	7.88 ± 2.27	10.48 ± 3.10	<0.001
Lymphocyte ($\times 10^9$/L)	3.07 ± 1.35	1.78 ± 0.54	1.13 ± 0.39	<0.001
Monocyte ($\times 10^9$/L)	0.53 ± 0.30	0.53 ± 0.35	0.54 ± 0.42	0.955
Hemoglobin (g/L)	146.21 ± 16.39	145.95 ± 17.64	145.75 ± 19.39	0.965
Platelet ($\times 10^9$/L)	215.67 ± 58.75	214.06 ± 59.87	212.38 ± 54.29	0.841
Temporary pacemaker	71 (33.5%)	87 (41.0%)	70 (33.0%)	0.155
Intra-aortic balloon pump	2 (0.94%)	4 (1.9%)	6 (2.8%)	0.361
Thrombus aspiration	50 (23.6%)	47 (22.2%)	50 (23.6%)	0.995
Infarct-related coronary artery				
Left anterior descending	107 (50.5%)	99 (46.7%)	113 (53.3%)	0.394
Left circumflex	21 (9.9%)	23 (10.8%)	26 (12.3%)	0.737
Right coronary artery	84 (39.6%)	93 (43.9%)	81 (38.2%)	0.466
Number of coronary arteries narrowed				
>1	155 (73.1%)	167 (78.8%)	162 (76.4%)	0.390
Use of medicine in hospital				
Aspirin	192 (90.6%)	189 (89.2%)	195 (92.0%)	0.608
Statin	197 (92.9%)	193 (91.0%)	201 (94.8%)	0.317
ACEI/ARB	109 (47.2%)	103 (48.6%)	115 (54.2%)	0.305
β-Blocker	122 (57.5%)	137 (64.6%)	136 (64.2%)	0.244
Spironolactone	172 (81.1%)	166 (78.4%)	173 (81.6%)	0.652
CTFC	16.17 ± 6.46	21.44 ± 11.25	25.04 ± 14.88	<0.001
CTFC ≥ 40	1 (0.5%)	9 (4.2%)	22 (10.4%)	<0.001
In-hospital MACE				
Arrhythmia	37 (17.5%)	42 (19.9%)	57 (26.9%)	0.049
Heart failure	38 (17.9%)	36 (17%)	42 (19.8%)	0.744
Angina	23 (10.8%)	35 (16.5%)	44 (20.8%)	0.020
In-hospital mortality	2 (0.9%)	5 (2.4%)	14 (6.6%)	0.003
Mortality out of hospital follow-up 12 months	7 (3.3%)	12 (5.7%)	24 (11.0%)	0.005

Data are expressed as mean ± SD for continuous variables or percentage (%) for categorical variables.
CK-MB, creatinine kinase-MB; hs-CRP, high sensitivity C-reactive protein; GFR, glomerular filtration rate; LVEF, left ventricular ejection fraction; HDL, high-density lipoprotein; LDL, low-density lipoprotein; WBC, white blood cells; ACEI, angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker; CTFC, corrected thrombolysis in myocardial infarction frame count; MACE, major adverse cardiovascular event.

59.22 ± 11.29 years, 76.9% men in tertile II, and mean age 59.13 ± 12.00 years, 77.8% men in tertile III, respectively. Age and gender were not statistically different across tertiles (p = 0.941 and p = 0.896, respectively). Besides, the duration from symptom onset to recanalization for group tertile I was 4.830 h (3.170–9.628), group tertile II was 5.500 h (4.000–9.500), and group tertile III was 6.460 h (4.500–10.628), respectively. There was no obviously significant difference among the three groups.

Correlation between NLR and the traditional clinical risk factors of myocardial infarction

As shown in Table 1, NLR, regardless of the use of invasive procedures or standard medical therapy, was observed to be correlated with several traditional clinical risk factors. Patients with higher NLRs were more likely to have increased hs-CRP, WBC, peak creatinine kinase-MB (CK-MB), and heart rates ($p < 0.001$, $p < 0.001$, $p < 0.001$, and $p < 0.001$, respectively), and decreased total cholesterol (TC), low-density lipoprotein (LDL) levels and systolic blood pressure (SBP) (p = 0.026, p = 0.003, and p = 0.048, respectively).

NLR is correlated with CTFC

Moreover, we observed that patients in tertile III had increased CTFC compared with those in tertile II and I [25.04 ± 14.88 (III), 21.44 ± 11.25 (II), 16.17 ± 6.46 (I), $p < 0.001$; Table 1]. Further analysis revealed that a positive correlation was found between CTFC and NLR (r = 0.517, $p < 0.001$, Table 2 and Fig. 1). CTFC was also correlated with glomerular filtration rate (GFR), WBC, neutrophil, lymphocyte, and NLR (p = 0.004, $p < 0.001$, $p < 0.001$, and $p < 0.001$, respectively; Table 2).

Table 2
Correlation between the baseline clinical data and CTFC as well as in-hospital mortality.

Variable	CTFC		in-hospital mortality	
	r	*p*-Value	*r*	*p*-Value
Age (years)	0.273	0.085	0.21	0.802
Systolic pressure on admission (mmHg)	−0.280	0.063	−0.316	0.004
Heart rate on admission (min^{-1})	−0.244	0.304	0.235	0.377
Left ventricular ejection fraction on admission (%)	−0.209	0.839	−0.28	0.045
Peak CK-MB (U/L)	0.257	0.184	0.205	0.895
Hs-CRP (mg/L)	0.268	0.111	0.294	0.018
GFR (mL/min/1.73 m^2)	−0.322	0.004	−0.334	0.001
Total cholesterol (mmol/L)	−0.267	0.119	−0.282	0.039
LDL (mmol/L)	−0.271	0.097	−0.297	0.015
WBC ($\times 10^9$/L)	0.357	<0.001	0.264	0.110
Neutrophil ($\times 10^9$/L)	0.458	<0.001	0.284	0.035
Lymphocyte ($\times 10^9$/L)	−0.418	<0.001	−0.323	0.002
NLR	0.517	<0.001	0.439	<0.001

CK-MB, creatinine kinase-MB; hs-CRP, high sensitivity C-reactive protein; GFR, glomerular filtration rate; LDL, low-density lipoprotein; WBC, white blood cells; NLR, neutrophil/lymphocyte ratio; CTFC, corrected thrombolysis in myocardial infarction frame count.

NLR is correlated with in-hospital and long-term mortality

Aside from CTFC, NLR was also strongly correlated with in-hospital and long-term mortality. There were 21 in-hospital deaths, and 43 deaths in total over the follow-up period. The causes for in-hospital deaths were complications after myocardial infarction and cardiac death, such as heart failure, malignant arrhythmias, and cardiac shock. The 43 late deaths after discharge were all-cause mortality. Correlation between NLR and in-hospital mortality was first analyzed and turned out to be strongly correlated with $r = 0.439$ and $p < 0.001$ (Table 2). Among patients who survived hospital discharge, 11.0% of mortality in the 12-month follow-up was observed in tertile III ($n = 24$), compared with 5.7% ($n = 12$) in tertile II, and 3.3% ($n = 7$) in tertile I, respectively ($p = 0.005$). The major separation between the Kaplan–Meier curves was observed during the third months of follow-up (Fig. 2). Positive trends between NLR and in-hospital and long-term mortality were supported by monotonic increases in the percentage of deaths from tertile I to tertile III ($p = 0.003$ and $p = 0.005$, respectively; Table 1).

NLR is an independent predictor of CTFC and in-hospital mortality

Variables associated with CTFC and mortality were analyzed using multivariate logistic regression (Tables 3 and 4). On multivariate regression, NLR (OR 2.031 per unit increase, 95% CI: 1.627–2.435, $p < 0.001$) and heart rate on admission (OR 0.932 per unit decrease, 95% CI: 0.878–0.986, $p = 0.017$) remained independent predictors of CTFC after PCI (Table 3). NLR (OR 1.176 per unit increase, 95% CI: 1.025–1.351, $p = 0.021$) was demonstrated as an independent predictor of in-hospital mortality (Table 4). Fig. 3 shows that threshold value of NLR was 5.9 (sensitivity: 63.7%, specificity: 61.1%) for predicting in-hospital mortality (AUC = 0.607, 95% CI: 0.475–0.739, $p = 0.253$). Taken together, NLR was shown to be an independent predictor of coronary blood flow and in-hospital mortality.

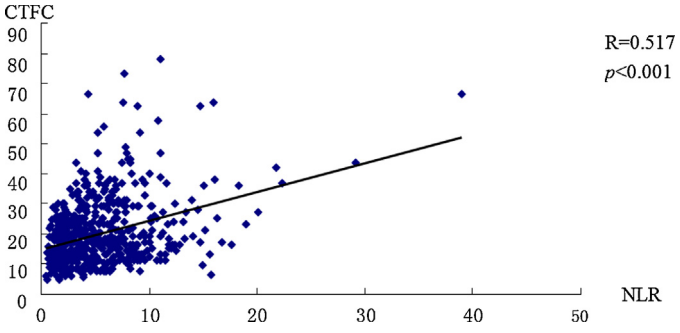

Fig. 1. Correlation between corrected thrombolysis in myocardial infarction frame count (CTFC) and neutrophil/lymphocyte ratio (NLR).

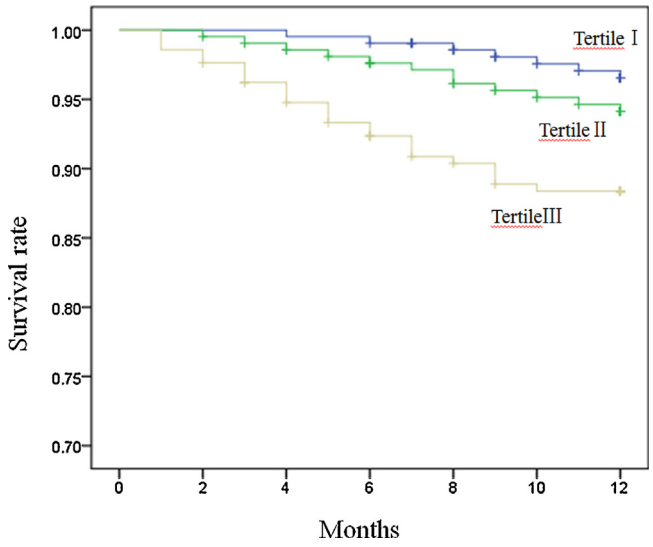

Fig. 2. Long-term survival rates among patients from different tertiles. Patients were categorized into three tertiles according to neutrophil/lymphocyte ratio upon admission (tertile I < 3.0; tertile II 3.0–6.40; tertile III > 6.40).

Discussion

The results of the current study indicate that: (1) NLR could predict coronary blood flow in patients with STEMI undergoing PCI; (2) NLR acted as a potent independent risk factor for in-hospital mortality; (3) NLR also appeared to have some implications for long-term mortality.

Inflammation has been reported to be involved in the initiation, development, and rupture of atherosclerotic plaque; therefore inflammation markers have been widely studied as predictors of CVDs. Leukocyte counts and traditional risk factors have been used in the diagnosis of coronary artery disease (CAD) [18]. Studies have also found that total WBC count acted as an independent risk factor for CAD [19,20]. Neutrophil and lymphocyte counts have also been studied as inflammation markers [21].

Of note, NLR, due to its potential as predictors for various diseases, has been received much attention in the field of CVD [8,16,22–24]. NLR reflects the balance between neutrophil and lymphocyte levels in the blood. Compared to other inflammatory markers, NLR is a simpler and less expensive diagnosis indicator. It has been observed that NLR demonstrated a certain value in the diagnosis and prognosis of acute coronary syndrome [25], angina [24], the severity of CAD [22,26], long-term prognosis in acute decompensated heart failure [27] as well as some other non-cardiac disorders [23,28]. Cho's group investigated the combination use of hemoglobin level and NLR for risk evaluation in patients with STEMI undergoing primary PCI. Their results showed that the combination of hemoglobin level and NLR could predict short-term clinical outcome. However, their study failed to show any correlation between NLR and coronary blood flow [29]. In our current study, by measuring CTFC and potentially related factors in blood, we demonstrated that NLR was an

Table 3
Effects of multiple variables on CTFC in univariate and multivariate logistic regression analysis.

Variable	Unadjusted OR	95% CI	*p*-Value	Adjusted OR	95% CI	*p*-Value
Systolic pressure on admission (mmHg)	0.977	0.940–1.014	0.222	0.996	0.960–1.032	0.820
Heart rate on admission (min^{-1})	0.974	0.919–1.029	0.347	0.932	0.878–0.986	0.017
Hs-CRP (mg/L)	1.023	0.997–1.048	0.086	1.002	0.976–1.027	0.876
Total cholesterol (mmol/L)	0.499	−0.381 to 1.378	0.266	0.914	−0.842 to 2.671	0.307
LDL (mmol/L)	0.454	−0.450 to 1.368	0.324	0.777	−1.050 to 2.604	0.404
WBC ($\times 10^9$/L)	1.821	1.500–2.143	<0.001	1.328	0.510–2.145	0.497
Neutrophil ($\times 10^9$/L)	2.741	2.423–3.059	<0.001	1.168	0.255–2.081	0.739
Lymphocyte ($\times 10^9$/L)	0.102	−0.743 to 0.947	0.813	0.408	−0.905 to 1.720	0.542
NLR	2.659	2.422–2.896	<0.001	2.031	1.627–2.435	<0.001

CTFC, corrected thrombolysis in myocardial infarction frame count; hs-CRP, high sensitivity C-reactive protein; LDL, low density lipoprotein; WBC, white blood cells; NLR, neutrophil/lymphocyte ratio; CI, confidence interval; OR, odds ratio.

Table 4
Effects of various variables on in-hospital deaths in univariate and multivariate logistic regression analysis.

Variable	Unadjusted OR	95% CI	*p*-Value	Adjusted OR	95% CI	*p*-Value
Systolic pressure on admission (mmHg)	0.974	0.957–0.992	0.005	0.977	0.957–0.998	0.029
Heart rate on admission (min^{-1})	1.006	0.984–1.028	0.613	0.981	0.960–1.002	0.075
Hs-CRP (mg/L)	1.009	1.002–1.015	0.012	1.005	0.994–1.016	0.406
Total cholesterol (mmol/L)	0.700	0.445–1.103	0.124	0.915	0.355–2.356	0.854
LDL (mmol/L)	0.661	0.417–1.049	0.079	0.585	0.215–1.591	0.294
WBC ($\times 10^9$/L)	1.098	0.976–1.236	0.118	1.068	0.697–1.637	0.763
Neutrophil ($\times 10^9$/L)	1.151	1.027–1.291	0.016	0.878	0.554–1.392	0.581
Lymphocyte ($\times 10^9$/L)	0.432	0.221–0.846	0.014	0.857	0.341–2.155	0.743
NLR	1.159	1.082–1.242	<0.001	1.176	1.025–1.351	0.021

Hs-CRP, high sensitivity C-reactive protein; LDL, low-density lipoprotein; WBC, white blood cells; NLR, neutrophil/lymphocyte ratio; CI, confidence interval; OR, odds ratio.

independent predictor for CTFC (coronary blood flow) in patients with STEMI undergoing primary PCI. Besides, our results also indicated that CTFC was correlated with GFR, WBC, neutrophil count and lymphocyte count.

There are also a few studies that investigated the value of NLR in predicting mortality in patients with CVD. Papa et al. conducted a 3-year follow-up study for patients who had CAD. Their study revealed that NLR was able to predict cardiac mortality in patients with stable CAD [15]. An 8-year follow-up study of STEMI patients accomplished by Shen et al. showed that there was a relation between NLR and long-term mortality. But their study measured the NLR in the first three days of STEMI, which could be affected by various factors during the in-hospital period [30]. Duffy and colleagues found out that pre-procedural NLR was correlated with increased risk of long-term mortality in patients undergoing primary PCI. However, they failed to specify the characteristics of the study population [11]. In our study, blood samples were collected immediately after admission for analysis of NLR, etc. and patients were well characterized. And our results revealed that NLR is an independent predictor for in-hospital mortality in patients with STEMI undergoing PCI.

Taken together, by analyzing the correlation between NLR with coronary blood flow and in-hospital as well as long-term mortality in 636 patients with STEMI undergoing PCI, our results showed that NLR was an independent risk factor for coronary blood flow as well as in-hospital mortality. Of note, further study, although beyond the scope of this study, is warranted to investigate the relationship between NLR and coronary blood flow and short-term and long-term mortality in patients with STEMI undergoing PCI based on a larger sample size from a multicenter database.

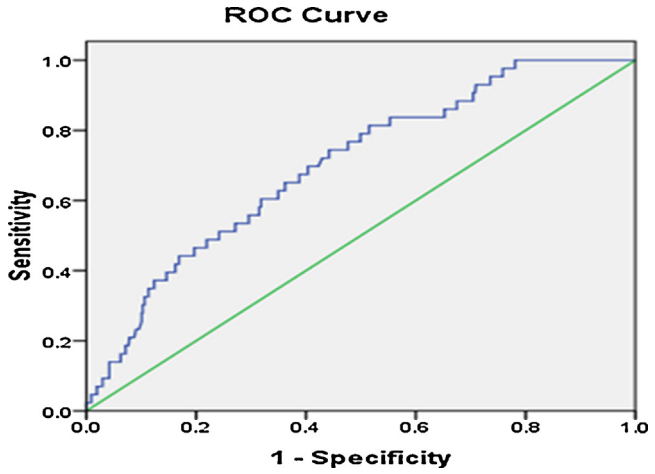

Fig. 3. Receiver operating characteristic (ROC) curves of neutrophil/lymphocyte ratio.

Conclusion

NLR, an indicator that can be tested in the laboratory with low cost and time consumption, is independently correlated to coronary blood flow and acts an independent risk factor for in-hospital mortality in patients with STEMI undergoing PCI.

Conflict of interest

The authors declare no conflicts of interest.

Acknowledgement

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